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ABSTRACT

A literature review was conducted to determine the meaning, symptoms, causes, and treatment of dyslexia. The review revealed that dyslexia simply means "distorted words," and is the inability to read either phonetically or visually. Among the symptoms of dyslexia disclosed by the review are motion sickness, retardation in reading of from 6 months to 2 years, bizarre spelling, and inattention to directions. The research reviewed indicates a number of causes for the problem ranging from prenatal influences to genetic factors and from birth damage to abnormal brain cells. Three methods of remediation are most often used in treating dyslectic students: the Frenald tracing method, which uses a sound-syllable approach; the Orton-Gillingham-Stillman method, which classifies sounds according to where they are formed in the mouth; and a combination of these approaches, which appears to be the most effective treatment. (FL)

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SOME SYMPTOMS, CAUSES, AND REMEDIATIONS OF DYSLEXIA

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May 1981

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SOME SYMPTOMS, CAUSES, AND REMEDIATIONS OF DYSLEXIA

I. INTRODUCTION AND DEFINITION OF DYSLEXIA

The entire conference of the Orton Society held in Seattle during November 1981 was devoted to a study of dyslexia. During the past decade, and especially in the past five years, an exceeding amount of attention has been directed toward those suffering from this elusive malady. Since the topic is one of current interest to psychologists, it is the purpose of this paper to look at the definitions of dyslexia, some symptoms of persons who have it, the research concerning some of its possible causes, and some current suggestions for its remediation.

Dyslexia means defective reading, while hearing and speaking remain relatively unimpaired, according to John Money (1962, pp. 9-10). Pirozzolo (1979, pp. ix, 7-9) notes that the syndrome of developmental dyslexia, so named because of a suspected lag in development, leads to unexpected reading failures. A research team from the World Federation of Neurology found that the malady is a disorder in learning to read even though the child has normal intelligence, good school instruction, and adequate socio-economic opportunities. Miles (1970, p. 13) says dyslexia is a variety of aphasia---the inability to handle and order abstract material. Simpson and Abromovitch (1980, p. 83) find that alexia is a total reading disability, but dyslexia is only a varying degree of impairment. Myklebust (1965, p. 126) calls it a disturbance of read language; while Cole (1979, p. 680) feels Orton's

definition of "specific reading disability" best explains the phenomenon. The brain and mind are normal, but the "processing of visual symbols is disturbed by visual-spatial confusion" in the dyslexic child. Finally, George Blache (1976, p. 179) notes that dyslexia actually means "distorted words." Originally it meant the person had inherited a lifelong inability to learn reading or spelling and was probably incurable. Even some educators felt the condition was irremediable.

The problem of defining dyslexia is further complicated when one tries to classify dyslexic children. They do not form a homogeneous group. As Simpson and Abromovitch (1980, p. 84) explain, the only way to classify dyslexic children is with a "shotgun approach." Perhaps Ellington was correct in saying that children are as handicapped by the "ignorance" surrounding dyslexia as by the dyslexia itself (Rome and Osman, 1979, p. 673). Some of this ignorance appeared as recently as August 1980 when the British Department of Education refused to recognize the existence of dyslexia or word-blindness. In a newspaper article, the deputy educational officer, John Fox, admonished Birmingham parents not to send their children to a dyslexic institute for help because the idea of dyslexia is against the policy of the British Department of Education (Hempel, 1980, p. 7).

In 1971 Broder found three sub-types of dyslexics: the dysphonetics who are non-phonetic, the dyseidetics who can not respond to words as a whole, and the alexics who have trouble with both phonics and whole words (Dalby, 1979, p. 241). These three types are almost synonymous with the auditory dyslexics, the visual dyslexics, and the auditory-visual dyslexics which will be discussed in the next chapter.

Originally some educators assumed that dyslexia was the result of English's non-phonetic spelling patterns. Evidence has since proved that dyslexia occurs in a variety of diverse languages including Swedish, Japanese, and even the ultra-phonetic Spanish (Pirozzolo, 1979, p. 8). Far from being an oddity of the English-speaking countries, dyslexia is a world problem which merits the attention of psychologists and researchers everywhere.

In summary, dyslexia is an inability to read phonetically, visually, or both. Some educators still deny its existence although it has been found around the world. The symptoms of dyslexia will be discussed in the next chapter.

II. SYMPTOMS OF DYSLEXIA

Children may have visual, auditory, or emotional problems which interfer with their ability to read without having dyslexia. To help differentiate the dyslexics from others who may have reading problems, this chapter will look at some of the symptoms which suggest dyslexia. Money (1962, p. 16) warns that no one has yet found a "foolproof" way of diagnosing the malady. The price of failure to diagnose it is so high, however, that one needs to try even if a foolproof method does not exist. The risk of diagnosing dyslexia and receiving help without having the problem would be infinitely preferable to the alternative which Rome and Osman (1979, pp. 660-1) offer. They mention the devastation dyslexia brings upon those who have failed to receive help in a literate society. Such unfortunates have been unable to cope with schoolwork since childhood, and they will continue to suffer the rest of their lives. To avoid such a possibility, Rome and Osman agree with Silver and Hagin that a child of normal intelligence who is seriously retarded in reading has a 90% chance of having a specific learning disability. They also agree with Dr. Bertil Soderling of Sweden who says that a child who cannot read and/or spell during the first three grades has dyslexia.

Before making a diagnosis, Myklebust (1973, p. 80) suggests that the child should see an opthalmologist, a neurologist, an electroencephalographist, a psychologist, and a reading specialist. The child's general health should also be considered in the diagnosis. Rome and

Osman (1979, pp. 661-2, 672) note that frequent headaches, stomach-aches, and excuses to remain out of school are often symptoms of a child struggling with a specific language disability. When hearing, vision, and general health prove normal, the assumption that the child is dyslexic can be confirmed with a 5-step program: 1. school history, 2. family history, 3. IQ estimate, 4. oral reading sample, and 5. spelling sample.

An educational history should include what subjects were easiest and hardest in school as well as the child's feeling about attendance. Often the child says he/she was sick and got behind. The parents say the child daydreams, appears lazy, disruptive, and inattentive. The teacher may describe the child as "casual" to protect himself/herself. Such a history strongly suggests dyslexia.

One should check the family history to see if other members had reading or spelling difficulties. It is unusual to find a true dyslexic without a family history of similar problems. Buckley (1981, p. 297) suggests asking if the child had motion illness, for Frank and Levinson found that anti-motion sickness medicine offered some help to dyslexics. Likewise Bettman et al (1972, pp. 101-2) studied 47 dyslexic children from ages 7-14. Forty-nine percent had a family history of dyslexia in their immediate background while only 6% of the 58 good readers had such problems. This is a highly statistically significant finding.

Speech patterns, language ability, and neuro-behavorial development help to estimate intelligence and to distinguish between dyslexia and mere backwardness is reading (Pirozzolo, 1979, p. 9). A child of superior IQ

who is doing only average work is not living up to his/her potential and may have dyslexia. Often the test-setting causes dyslexics to do poorly because they expect to fail. The doctor should evaluate the child's conversation and how well he/she copes in a non-academic setting as well as mere test scores to determine intelligence (Rome and Osman, 1979, p. 662). Paine (1972, p. 69) finds that the typical child with brain dysfunction does learn to read, but the true dyslexic does not---even when provided with the best remedial teaching, the disability persists.

To obtain the sample of oral reading, Rome and Osman (1979, pp. 662-3) suggest using Gray's Oral Reading Paragraphs. No grade needs to be given---a qualitative analysis is adequate. Likewise a short, standardized list of spelling words plus a short paragraph on a familiar subject like "My Pet" will give a good idea of the reversals and spelling problems. The doctor can also assess the amount of time required for the child to do the written work.

Miles (1970, pp. 1-3) suggests the need to look for a special weakness in reading or spelling or both. If the child's failures are puzzling, make no sense, it may be dyslexia. Myklebust (1973, p. 128) finds the ability to syllabicate is the highest discriminator between good and poor learners while comprehension and vocabulary are the most critical needs of poor learners.

Critchley (1970, pp. 118-121) says that dyslexics can be taught to read and says if they are intelligent enough in other areas, they can succeed in college or even graduate school in spite of their handicap. Harris (1979, p. 342) cautions that the possibility of outgrowing this deficit remains

uncertain, for the defect may be a permanent one. Dyslexics often have to learn coping techniques to adjust to their problem.

Buckley (1981, pp. 296-7) thinks one of the symptoms of dyslexics is their habit of reversing letters and reading a passage over and over to make sense from it. Such students are punished at home and at school for not wanting to learn when their real trouble is a learning problem. Beauvois and Derouesne (1981, pp. 21-2) find that patients with "surface dyslexia" cannot read words spelled irregularly, but can read regularly spelled nonsense syllables. Patients with "deep dyslexia" or phonemic dyslexia are incapable of reading non-words but can read all kinds of real words. These latter patients have lost the phonetic process, but retain reading. Again these two groups are similar to the visual and auditory dyslexics. The first group can learn regular phonetic syllables, but not unphonetic ones; while the latter group can remember visual words but not phonetic syllables.

Golden (1980, p. 10) reports a test of tracking lights, requiring no reading, which were used with fast, normal, backward, and dyslexic children. The fast, normal, and backward children made similar scores, but the dyslexics showed erratic eye-movements. Such eye-movements may be a symptom of dyslexia.

Sobotka and May in 1977 found children were dyslexic if they read only six months below grade level (Pirozzolo, 1979, p. 9). In contrast, Taylor, Satz, and Friel (1979, pp. 84, 87, 98-9) feel that dyslexics cannot be distinguished from other poor readers and educators need to revise their concept of dyslexia. They do admit that some children might inherit

dyslexia from their parents. These views seem to offer the two extremes: all children six months behind in reading have a degree of dyslexia as opposed to almost no children have it.

One significant finding of Beauvois and Derouesne (1981, p. 36) shows that a totally different process is used in word recognition from that used in reproducing a word on paper---spelling. Jordan (1972, p. 4) noted this fact when he found that some dyslexics lacked encoding---the ability to translate language into written words---while others were unable to translate symbols into spoken words. Duane (1979a, p. 364) also found that spelling disorders are frequently associated with dyslexia.

Miles (1970, pp. 3, 6-9) thinks one of the best clues to dyslexic children over age nine or ten is the confusion of "b," "d," "p," and "q." This twisting of symbols is called strephosymbolism. Often the dyslexic cannot see the difference between his/her misspelled words and the correct spellings---in short, the child has a form of word-blindness. Such children may resort to watching mouth movements and spelling phonetically to compensate for their disability. The result is phonetic spellings and the mixing of "b" and "p" which have similar mouth movements. Often dyslexics may remember the word in one instance and forget it in the next. Bizarre spelling and reversals go together; however, reading improves sooner than spelling. Children whose spelling and performance fail to reach their intellectual potential probably are suffering from dyslexia and need special attention.

Frank and Levinson in 1976 flashed speeded letters onto a screen. The dyslexics saw the letters as a blur much faster than the good readers.

The researchers concluded that this was a vestibulo-cerebellar symptom of dyslexia (Pirozzolo, 1979, p. 26).

Eileen Simpson (1979, p. 217), who has dyslexia, feels there is no cure for it. She is never symptom-free, but she can manage her handicap. On good days she can spell reasonably well, but she always has trouble proofreading, map reading, and not transposing digits in a phone book. Fortunately, she has a good memory and can function.

Sobotka et al (1977, p. 366) found that dyslexic children performed differently from normals on performance measures as well as on verbal ones. Perhaps more than just the reading area is involved in dyslexia.

Pirozzolo (1979, p. 33) attempts to show that there are two types of dyslexia: auditory-linguistic and visual-spatial. Duane (1979a, p. 638) says Broder felt an analysis of spelling would differentiate between those with auditory processing difficulties and those with visual ones. Myklebust (1965, pp. 29-30) tells of a boy who could not associate sounds with letters but could write words correctly when the letters were dictated to him or he copied the words visually. Suffering from auditory dyslexia, he showed brain dysfunction in neurological and electroencephalographic studies. If a child does not have both the auditory and visual aspects of letters, he cannot learn to read normally. Auditory-linguistic dyslexics have impaired language, lower verbal IQ's, and faulty grapheme-to-phoneme matching. They are also impaired on tasks requiring rapid linguistic processing.

Kenura found if two messages are presented---one to each ear, the right ear will be more reliable for language while music is a left-ear function (Dalby, 1979, pp. 250-1). Presumably this is because each ear

sends its message to the opposite hemisphere of the brain. It may be significant that most poor readers make phonetic rather than visual errors; thus English-speaking dyslexics easily learn to read Chinese characters which require a visual-holistic processing. Money (1962, pp. 21-2) says Kanji ideographs were easier to learn than phonetic-type Kana in Japan.

Pirozzolo (1979, pp. 86-7) finds that visual-spatial dyslexics showed directional disorientation, spatial disgraphia, dyscalculia, and finger agnosia (loss of feeling). Showing faulty left-to-right eye progression and return-sweep eye movements, they were impaired in tasks of visual discrimination, analysis, and memory.

Some have estimated dyslexics exist as low as 1 in 500 and others as high as 1 in 3 (Pirozzolo, 1979, p. ix). Jordan (1972, p. 4) feels few dyslexics have only one type of malady---most have two or more kinds of perceptual loss. Vogel (1975, pp. 79-84) mentions various research on the subsets in dyslexia. The first group were immature with perceptual problems and a higher verbal than performance score on the WISC. The second group included speech problems and poor language development with higher performance than verbal scores. A third group, which is the most severely dyslexic, had trouble with both auditory and visual learning. Following her own research, Vogel concludes that dyslexics differ from normal children in syntax ability, and even retention in their grade does not close the gap between the dyslexics and the normals younger than themselves. Perhaps remediation might help, but this is not a proven fact; however, the dyslexic's poor syntactic background may mean that he/she is more severely handicapped than previously thought.

Handedness may be somewhat associated with reading problems. Orton said that a suspiciously high number of children who had not developed handedness or were left-handed were among those who had exceptional reading problems (Pirozzolo, 1979, pp. 12-13). In a right-handed person, rightward gaze and head-turning occur in left hemisphere activities while right hemisphere acts are directed by a leftward gaze and head-turning. Dalby (1979, p. 252) says some researchers think this is a valid predictor of brain hemisphere functioning. Schwartz (1980, p. 97) thinks 95% of right-handers have the left hemisphere dominant for both language and motor activities while only 50% of left-handers find this true. Instead, they tend to be ambidexterous and rarely have consistent preferences. Dyslexic children have trouble with left-right discrimination and may be poorly coordinated or clumsy (Buckley, 1981, p. 296).

Sobotka et al (1977, p. 307) note that the Bender-Gestalt discriminated well at age 7 but was not strong in discriminating reading disability at ages 9, 11, or 13, although other studies found it did discriminate at ages 8 and 10. Rourke and Orr in 1977 found initially that visual-motor performance discriminated between the normal and retarded readers. Four years later these children could only be differentiated by problem solving, hypothesis testing, and abstract reasoning. The distinguishing factors had shifted from motor to language functioning. Perhaps a monitoring of regional cerebral blood flow would tell if the mental activities of dyslexics are different from those of normals (Dalby, 1979, pp. 247-53).

Buckley (1981, p. 300) says the dyslexic child has a disorder in the way incoming sensory data is organized for presentation to the brain.

The patterns involving balance, equilibrum, and coordination are involved in this disorder. Rome and Osman (1979, p. 673) warn that dyslexics often turn their frustrations upon themselves in depression, anxiety, and disturbance, for many dyslexics become juvenile deliquents.

Golden (1980, p. 10) reports that children go through a spontaneous recovery period about 11-12 years of age. The brain cells were not destroyed; they were just lazy. With new stimulus, they begin working, and children's reading often improves. This knowledge should help teachers to understand children better.

In summary, the dyslexic child may have had a history of motion sickness, school reading retardation ranging from six months to two years below grade level, and a variety of spelling problems. Dyslexia usually breaks down into two large categories---auditory-linguistic problems and visual-spatial ones. Many have only one type of problem while others have both. Some hope is offered when a spontaneous improvement occurs at ages 11 or 12, but usually the dyslexics will have a difficult school experience and trouble with reading and/or spelling throughout their lifetime.

III. CAUSES OF DYSLEXIA

Having looked at some of the symptoms of dyslexia, one needs to view the possible causes. Heterogeneity of guesses might be the best description of the basic for dyslexia. No one has an absolute explanation for the real cause, but a few ideas are persuasive.

Golden (1980, p. 10) reports that dyslexia is not due to environment or intelligence. In recent years the idea that emotional conflicts were the primary cause of language disabilities has proven to be at variance with the facts (Rome and Osman, 1979, p. 660).

Kawi and Pasamanick in 1959 found a statistical relationship between abnormal pregnancies and reading disorders (Money, 1962, p. 14). Bettman et al (1972, pp. 101-2) say that 24% of the 47 dyslexics tested had premature or pregnancy-birth problems as against 4% of the good readers. Such a comparison was highly significant statistically. Dalby (1979, pp. 243, 247) says fetal anomia, smoking mothers, and premature births are all associated with dyslexia. Some arguments have been presented, but more studies support the idea than reject it. Likewise neurological dysfunction in the newborn period was related to later learning problems.

Simpson and Abromovitch (1980, p. 84) note that some have suggested a genetic factor for dyslexia. Since genetic characteristics remain for a lifetime, Goldberg and Schiffman determined the problem was genetic; however, the characteristics of familial dyslexia are elusive or else more complex than initially thought. Further study should be made of dyslexic parents

who do and do not have dyslexic offspring (Dalby, 1979, pp. 244-6). Pirozzolo (1979, p. 12) agrees that reading difficulties run in families. Once thought to be a recessive gene, it was found to be dominant in a Swedish study where 88% of the 276 dyslexic cases showed other family members also suffered from dyslexia. Zangwill in 1962 found that a group of pure visual dyslexics were genetically determined (Vogel, 1975, p. 5). Likewise Clarke (1973, p. 140) includes family histories of stutterers, non-readers, and poor spellers as a sign of genetic dyslexia.

Levine (1979, p. 628) tells of a family with 5 boys and 2 girls where all the boys and their father had dyslexia, but the girls did not. He thought he had made a discovery until he found that Stevenson had written on this subject in 1907. Duane (1979b, p. 642) offers no explanation for the 3-5 fold higher reading retardation in boys than in girls, but says dyslexia usually affects males more than females. One explanation mentioned by Dalby (1979, p. 246) says children use both ears when they are very young, but third grade girls require a right-ear advantage to be good readers. By age 11 boys also require this right-ear advantage in order to read well. The study suggests that girls mature their reading skills at a younger age than boys.

Dr. James J. A. Cavanaugh thinks premature graying is an indicator of dyslexia. His views agree with Joyasekara and Street who found a greater evidence of dyslexia among children of older parents. They hypothesize that aging cells might be a factor (Dalby, 1979, p. 244).

The idea of a developmental lag as a cause for dyslexia has been one of the major explanations for the malady. Orton began this view by

saying dyslexia was a lack of maturity in all language skills (Vogel, 1975, p. 3). Pirozzolo (1979, p. 10) feels that learning lags and/or physiological immaturity cause dyslexia. Jordan (1972, p. 2) says many researchers think dyslexia is the result of lazy or underdeveloped centers in the brain that require over-teaching of specific skills like spelling, phonics, handwriting, etc. The "miraculous" recoveries seen in some children at ages 11-12 when myelination in the brain is fully developed support the maturation theory. Sobotka et al (1977, pp. 366-7) also note that perceptual motor differences decrease with age; therefore, they suspect a maturational lag occurs in the perception area. Miles (1970, p. 11) summarizes the maturational theory when he says one should talk less about brain damage and more about a failure in development.

Pringle Morgan in 1896 found a 14-year-old boy with "congenital word-blindness." Morgan suggested the child's angular gyrus was underdeveloped, and his hypothesis is still felt plausible although the theory has no neurological support. Yakovlev and Lecours in 1976 showed that the angular gyrus was one of the last regions of the brain to reach maturity. Since myelination speeds electrical impulses down the axons and insulates them from the noises of the neurons, it is plausible that late myelination may cause dyslexia. Perhaps late maturation of the fiber pathways in the left hemisphere causes auditory-linguistic dyslexia (Pirozzolo, 1979, pp. 7, 85-6). Dalby (1979, pp. 240, 256) says some researchers reject the maturational "lag" hypothesis because a lag suggests the child can catch-up eventually. Such catch-up may never occur.

The mixed dominance (left-handedness in a right-eyed person or vice versa) hypothesis of Orton appeared after he could not find brain pathology. Gesell and Anatruda in 1941 felt that when mixed dominance is present with language or reading disabilities, it represents a neurological deficit. This idea is still in vogue (Harris, 1979, p. 338).

Pirozzolo (1979, pp. 1-2) says neuropsychology is vitally interested in reading and reading disabilities. Researchers are continually looking for the biological cause for brain dysfunction and dyslexia. Pierre Paul Broca, a French neurologist of the last century, was the first to say that a language-motor disorder was caused by damage to the front of the inferior frontal gyrus in the left cerebral hemisphere. Phrenologists had thought specific functions were localized in the brain, but it was Broca's discovery of the language portion of the brain that gave credience to their work. Some have disagreed with Broca, but many neuro-scientists today feel a relationship exists between Broca's aphasia and the inferior frontal gyrus. Carl Wernick in 1874 added the knowledge that lesions in the posterior third of the first temporal gyrus in the left hemisphere caused aphasia in phonemic hearing.

Hinshelwood in 1917 felt one type of dyslexia was the result of a defect in the angular gyrus. Orton in 1937 broadened the task to writing and speech, but said they were physiological defects not brain damage (Vogel, 1975, p. 3). Pringle Morgan in 1896 and Hinshelwood in 1895 were among the first to recognize the relationship between brain dysfunction and reading disabilities. Although controversies have arisen, gradually educators are becoming aware of the value of these men's observations (Myklebust, 1973, p. 171).

Prenatal neurological damage affects the whole brain; therefore, it is the major cause of dyslexia according to Dalby (1979, p. 244). Buckley (1981, p. 290) suggests that since an obstruction of the umbilical cord in monkeys caused lesions resulting in crebral palsy, skull compression at birth may cause problems in temporal lobe functions during life. Duane (1979b, p. 647) agrees there probably are central nervous system involvements in dyslexia, but says they remain elusive. Buckley (1981, p. 289) notes that the neurological basis for dyslexia has been hesitantly accepted in the past twenty years. The term minimal brain dysfunction is bad because it sounds small and unimportant. Subtle dysfunction would be a better term. Duane (1979a, p. 634) explains that "soft" signs of central nervous system dysfunction are soft only because they do not appear to all examiners on a consistent basis.

Abrams in 1968 found three neurological dysfunctions in severe reading disabilities: 1. diffuse brain damage, 2. lesions in the occipital-parietal area, and 3. a disturbed pattern of neurological organization without definite damage (Vogel, 1975, p. 2).

Pirozzolo (1979, pp. ix, 4-5) says that research is quite sound which strongly suggests a neurological basis for developmental dyslexia. Pure word-blindness is the ability to write, but not to read. Dejeune said lesions interrupted the connection between the angular gyrus and the primary visual area or word center. A second lesion in the corpus callosum disconnected the right visual cortical area from the left hemisphere and made the person word-blind. Some disagreed with Dejeune, but his conclusions have been supported by many investigators. Dalby (1979, p. 257) says there is sufficient

evidence to associate atypical brain functioning with the source of reading difficulties in children of normal intelligence.

A news item concerning Duffy, Galaburda, and Geschwind (1980, p. 1) says that 80-90% of children with learning disabilities who attended clinics in reading were found to have unexpected functional abnormalities in their frontal brain lobes.

George Pavilidis at Manchester University claims dyslexia is caused by a brain malfunction which directly affects the eye-movement system. He hopes to develop a simple reliable test for early diagnosis (Golden, 1980, p. 10). Likewise Pirozzolo (1979, pp. 32-3) and Reyner found a relationship between reading eye-movements and dyslexia. In 1978 they found that auditory-linguistic dyslexics reacted like young children first learning to read with many fixations per line, longer fixations, and many regressions; however, they could read easy material. The researchers suggested that the dyslexia caused the lateness of the eye-movement development instead of vice versa--- a rather unique hypothesis. Visio-spatial dyslexics had unusual eye-movements for they scanned a line leftward and used regressions. Pirozzolo admitted this problem might be a spatial disorder rather than one of eye problems.

Dr. Julio de Quiros found that 52 out of 63 learningly disabled children had abnormal responses to an inner ear test. He also observed the connection between the vestibular pathways and the oculomotor nerves which control eye-movements and provide feedback for the fine muscles in writing. Buckley (1981, pp. 296-7) concluded that the overlap of distractibility and dyslexia may be the result of two kinds of dyslexia--- the first type is a dysfunction of the vestibulo-cerebellar tracking and

orienting circuits; the second form results when the ergotrophic pathways of the limbic system fail to provide proper input. Formerly dyslexia was thought to be of cerebral origin. Research now has found that it can be a vestibulo-cerebellar interference which prevents the integration of both auditory and visual sensory input. Buckley thinks these "brain stem circuits are crucially involved in the dyslexic disorder." Eileen Simpson (1979, p. 231) reports that the involvement of the cerebellum was first suggested by Drs. Jan Frank and Harold N. Levinson of New York Downstate Hospital. Duane (1979b, p. 646) raises a negative voice to this cerebellar theory. While admitting that seasickness medicine is supposed to help, he finds no proof for this theory's merit nor for its remedy.

Dalby (1979, p. 253) notes that 10 out of 24 right-handed dyslexics had a reversal of the asymmetry of the right parieto-occipital region. This area was wider on the right side than on the normal left. Although he did not see it as a cause of dyslexia, he felt it might be a factor.

Another possible cause of dyslexia is brain damage. Neo-natal split-brain kittens took longer to learn experimental tasks than normal kittens, but both groups learned faster than the normals when given amaphetamines. The corpus callosum may be a central programming device whose late development may cause minimal brain dysfunction just as a late development of the angular gyrus may cause dyslexia.

Organic brain lesions have long been accepted as a cause for epilepsy, cerebral palsy, and gross retardation. It is a hard sign on the EEG, for it is not as vague or transitory as the neurologically "soft" signs (Buckley, 1981, p. 289). Dalby (1979, pp. 252-3) says a relationship

exists between certain biochemical abnormalities in the brain and dyslexics with abnormal EEG's. Although some normal children did have abnormal EEG's, newer methods of testing may change that. Park found that many dyslexics who did not have brain injury suffered from thyroid and pituitary dysfunctions; neurological problems; respitory, heart, and blood disease; poor vision; and behavorial problems. He felt their glandular problems, especially hyperthyroidism, might account for their reading difficulties (Smith and Carrigan, 1959, p. 12).

With the exception of a specific post-natal brain injury, all of the foregoing causes including a malformed or malfunctioning angular gyrus, impaired cerebral dominance, delayed cerebral maturation, cerebral biochemical imbalance, and genetic factors may be only indicators of a deeper underlying problem. The next hypothesis appears to this writer to offer the best solution to the dyslexia puzzle, for it can incorporate all the fragmentary findings into one underlying cause.

Pirozzolo (1979, p. ix) mentions an autopsy on a dyslexic child who died of a vascular malformation in 1968. The report mentioned "an atypical cortical gyral pattern with microgyria and pachygyria as well as an absence of the typical pattern of six well-differentiated layers of cortex." This single case could not prove a cause for dyslexia. However, more recent findings are reported in an article concerning Duffy, Galaburda, and Geschwind (1980, p. 1). Neurologist Dr. Anthony Galaburda also found abnormal cells in an autopsy on a twenty-year-old dyslexic killed in a car accident. The abnormal cells found in the language area of the cortex were similar to those producing epilepsy; in fact, Galaburda says dyslexia

could be an epileptic phenomenon. The brain appeared normal in an X-ray. Only when a sliced section was placed under a microscope did the abnormal cells appear. They were not borderline differences, but striking ones which were formed prenatally with the brain's development. The finding is a landmark one made possible by cyoarchitectonics---a study of the way cells are arranged in the brain's cortex. The writer finds this explanation plausible because unlike brain damage, this is a birth defect which may be genetically inherited. Abnormal cells in a part of the brain could also explain the dysfunction or erratic functioning of other brain areas. All of the various sections of the brain which seemed late in maturing or were malfunctioning could trace their problems to this underlying birth defect.

The writer has found some evidence to support the connection between epilepsy and dyslexia. At present a boy suffering from auditory dyslexia is her pupil. He has a younger brother and an uncle who both suffer severe seizures frequently. It is possible, even highly probable, that the boy also has abnormal brain cells which are causing his reading problems. Another student who was hyperactive and had an abnormal EEG, also had an epileptic brother and a mother who suffered from migrane headaches. His mother's brother had severe learning problems in reading. Clarke (1973, p. 24) mentions that both she and her severely dyslexic son suffered from headaches after age eight. She also noted that she had periods of proficiency in articulation---times when she was not bothered with dyslexic mispronunciation of words---followed by migranes so severe that they could not be controlled by any medicine. Although these cases are few in number, they offer surprising similarities. Could abnormal brain cells be the basis for both

epilepsy and dyslexia? Galaburda and his associates seem to think so. They are now working on a new technique BEAM (Brain Electrical Activity Mapping) which uses electrodes painlessly attached to the head to determine abnormal brain functions. With this new tool, dyslexics may be identified earlier and given the remediation, or medication, they need before they become totally frustrated in a literate society.

In summary, this chapter has looked at some of the possible causes for dyslexia. Ranging from slow maturation to brain damage, the hypotheses are as varied as their authors. This writer favors the most recent findings which link the cause with an underlying birth defect--- abnormal cells in the brain's cortex. Such cells could account for most, if not all, of the diverse physical abnormalities and the genetic implications which the other theories attempt to explain. It is now time to turn from the suggested causes to possible remediations which will be discussed in the next chapter.

IV. REMEDIATION OF DYSLEXIA

Jordan (1972, p. 3) feels that the immediate concern of teachers should not be what caused this problem, but what to do about it. Duffy, Galaburda, and Gerschwind (1980, p. 1) say the most important aspect of dyslexia is early detection. When the child really can't read, it is too late for remediation. Jordan (1972, p. 9) finds that those dyslexics caught before grade three have an 80% chance of remediation; those caught before grade five have a 40% chance; but those who are left until grade seven have only a 5.7% chance of reaching grade level proficiency in encoding and decoding.

Rome and Osman (1979, pp. 660-671) who deal specifically with dyslexics in their reading clinic at Rochester, Minn., feel that to diagnose dyslexia and not remediate it is like diagnosing appendicitis without planning for surgery. Some children find reading as easy as carrying a feather while the dyslexic may find it is like carrying a ton. The remedial program may be titled learning disabilities, language disabilities, or perceptual problems---its title is unimportant so long as it meets the needs of the dyslexic. Unfortunately, most remedial reading classes are NOT designed for dyslexics. Even a teaching degree in learning disabilities does not insure the teachers can work with dyslexics and over-teach the material in a patient, unhurried manner. Adult education courses are seldom beneficial to dyslexics because they, too, use conventional methods,

not special ones. Unless helped, however, the dyslexics will suffer from the first introduction of the abc's until the end of their lives. Cole (1979, p. 677) warns that dyslexics need help from teachers trained and experienced in their type of education. Critchley (1970, p. 115) suggests that special teachers go from school to school to help the dyslexic students.

A few authors like Pirozzolo (1979, p. 87) and Jordan (1972, p. 2) feel that most types of dyslexia can be treated in the normal classroom if the teacher would make the adjustment because it is not the method, but the psychological contact between teacher and student that is the basis for remedial learning. Jordan does admit that brain damage should be treated with medication and possibly physical activity outside the regular classroom. Probably the regular homeroom is best, but only if the teacher is trained to meet the needs of the dyslexic child.

Levine (1979, p. 629) tried a number of interventions with dyslexics. Summarizing his treatments, he says that he tried Dexadrine and Ritlin which helped the hyperactive children; he tried eye-exercises with some and vitamins with others; he excluded sugar and food additives with another group; and he even had one child learn to creep---none of the dyslexics were helped! Pirozzolo (1979, p. 11) adds that even the Feingold bio-chemical diet showed no effect on school behavior or learning of children with minimal brain dysfunction.

Remediation for the dyslexic will seemingly have to come from a special reading program. Schiffman (1966, p. 246) says five main planks compose corrective reading: 1. parent education, 2. teacher education, 3. small group instruction, 4. individual small-group testing, and 5.

remediation of auditory or visual-perception and sequencing deficits. To achieve optimum instruction, Cole (1979, p. 677) suggests a well-structured program in a setting offering a minimum of auditory and visual distractions.

Rome and Osman (1979, pp. 666-671) note that a structured program moves essential learning from simple to complex. It teaches new material until it is thoroughly mastered, and it teaches the relationship of the new material to the old. Styles of teaching may vary, but they are good if they contain the essentials of a dyslexic program. Such a program would have visual drill of the sounds matched to cards; auditory drill when the child can blend two sounds; and finally, much repetition until the dyslexic can automatically retrieve sounds, rules, and patterns. The 4-R's of a remedial program are: 1. recognition for reading, 2. recall for spelling, 3. reproduction for writing, and 4. repetition for learning.

Eileen Simpson (1979, pp. 209-10) says to read aloud to the dyslexic. Start the book on the child's level, let him/her read; then read aloud again when he/she lays the book down. This only works if the parents enjoy reading aloud, and if they are not also dyslexic.

Jordan (1972, pp. 4-9) warns that most children with dyslexia have more than one type. Visual and auditory are often found together, but only one can be remediated at a time, or they will cancel each other out. Visual dyslexics are the easiest to remediate. If they have strong auditory skills, they can learn phonics and drill until they can decode syllables easily. Often visual dyslexics see letters backward. They need to read the words aloud to "hear" what they are decoding. Teaching left-to-right and top-to-

bottom progression is essential. Teachers should emphasize doing the job right the first time. Careful teaching of handwriting can eliminate some reading problems, but letting the child write incorrectly helps to fix misspelled words in his/her memory (Enstrom and Enstrom, 1970, pp. 27-30, 34). Money (1962, pp. 24-5) says acquiring English words is a feat of visual memory. The dyslexic needs a combination of visual and phonetic recognition in order to read. If the dyslexics have poor memories, they need to learn the reasons or rules for things (Cole, 1979, p. 678).

Since visual dyslexics often get confused with the sequences of days and months, the teacher should teach chronological and alphabetical sequencing. Over-teaching, drilling, and keeping track of pupil progress are needed (Jordan, 1972, pp. 5, 6, 71-82). Dyslexics may have trouble with various word endings, but Miles and Miles (1975, p. 51) feel that calling attention to them will encourage the child to look at the word in detail and break it down into manageable parts. The dyslexic must learn to synthesize and analyze---take apart and put together. The amount necessary will depend on the severity of the problem (Rome and Osman, 1979, p. 670). Jordan (1972, pp. 67-70) urges the teacher to find what the child needs and begin with the abc's if necessary. The teacher should relax and move at a slow pace keeping the work simple and highly structured for learning. The teacher's expectations of success can mean the difference between triumph and disaster for the visual dyslexic. Mann et al (1979, pp. 167-8) suggest remediations for visual-perception difficulties including: sorting items; matching concrete items; telling how two items are alike and different; matching pictures; and discriminating first cut-out shapes, then letters, words, and finally numbers.

Gardner (1974, pp. 138-9) says the most effective method for remediating developmental dyslexia is learning thirty Chinese characters matched to English words. Read from left-to-right, they form English sentences. Gardner was the only author who urged a strictly sight-word approach; all the others suggested phonics; however, Gardner's method is somewhat like the rebus reading used in some Learning Impaired classrooms. Jay Isgur has claimed success in teaching the alphabet with "m" shaped mittens or a "p" shaped pen, according to Gardner. The meaning and object help recall the shape. This writer also found that some children cannot recall the abstract names of the alphabet letters. In desperation she wrote rhymes such as "This umbrella handle went out on a rainy day; it's name is 'j.'" Such clues did prove effective in helping all the children to recall their letters.

Jordan (1972, pp. 6-8, 31-4) says the most difficult dyslexia to remediate is auditory because it is the inability to separate sounds of spoken language. It is similar to tone deafness in music, and may remain undiscovered until third grade if a sight-word approach to reading is used. It is usually the cause of poor spelling. One of the surest signs of auditory dyslexia is chronic erasing, crossing out, and rewriting. As a rule, these dyslexics must find their own sight-memory system for spelling. Some can learn through word families and then memorize enough to pass their grade. Four patterns of spelling characterize the auditory dyslexic: 1. transposed consonants, 2. phonetic spelling, 3. sound units omitted, and 4. sound units added.

Rome and Osman (1979, pp. 664-8, 672) think auditory dyslexics must learn the phonetic units of the language in order to succeed in reading and writing. Hearing themselves pronounce words may also give remediation. If the teacher explains the curve of hearing from tone deafness to absolute pitch, the dyslexics will understand their problem better. Dyslexia can also interfer with a child's auditory sequencing. Often these children appear as daydreamers or unmotivated when they are actually learningly impaired. They may even have trouble understanding what the teacher said to do first, next, and last in sequential order. Such children must learn all the phonetic units needed to write each sound. For example, they need to know that "tch" comes only after a short vowel; otherwise, one uses "ch." Rules are very important for dyslexics because they need a feeling of security. Taping lectures and taking notes later from the tapes is another good coping device.

Jordan (1972, pp. 86-107) suggests replacing all look-and-say words with phonics. He encourages progression by easy steps with much visual reinforcement and intense individual teaching of no more than 30 minutes at a time. He also mentions a utility corner with much practice, memory clues, consistent spelling rules, and even fun things to read like boxtops and labels.

Mann et al (1979, pp. 158-9) urge listening to very different sounds in nature; at home; in school; and finally to musical instruments, words, and letter sounds. Miles and Miles (1975, pp. 21-33) suggest that younger children need to learn consonant sounds before beginning spelling. After the consonants come the short vowels, and then the digraphs like "sh" and "ch." Dyslexics may not know the alphabet, but should learn the first five and last five

letters. They can then find the rest in the dictionary toward the middle. Rules concerning the use of the hard and soft "g" and "c" help. Both are hard before "a," "c," and "u;" while both are soft before "e," "i," and "y."

Older dyslexics need to begin with a fresh start. They should learn to type their material to make learning easier. Rome and Osman (1979, pp. 670-1) feel that the approach for adolescents should be different from that used with small children. They need to learn prefixes not individual letters. Harris (1979, p. 342) tells of one very new method which sent reading instruction to the right ear and pop music to the left while vision was blocked out from the left eye---thus all input went directly into the left hemisphere of the brain. This approach offered great improvement in learning, but either visual or auditory alone did so help.

Mazurkiewicz (1966, p. 173) urges using an initial teaching alphabet of regular phonetic spellings with dyslexics. The problem came later when these learners had to change to traditional reading. They found that their initial learning had been practically a waste of time.

Vernon, Cooley, and DuBois (1980, pp. 216-17) found deaf children who could finger spell, but could not master oral language. This finger method was taught to dyslexics as a whole-word method and seemed to work quite well. The dyslexic would say the word and finger spell it at the same time.

Miles (1970, pp. 14-17) thinks one of the big teaching problems with bright dyslexics is that a normal class is too hard, and a backward class is unsuitable. Some parents can help their children, others cannot. A tutor may be necessary; therapy usually lasts for 1-2 years. Miles and

Miles (1975, pp. 6-11) say that bright children are often hurt wondering if they are stupid or mad because they cannot do what others do, and the prognosis is not good, for they may be slow readers and poor spellers all their lives. Parents, too, are often made to feel guilty about having a dyslexic child. This is cruel and unfair. Both parents and child are usually frustrated, and the teacher or tutor needs to have a great deal of sympathy.

Cole (1979, pp. 679-80) says there is a need for a coordinated program involving the physician, the psychologist, the school administrator, and the teacher to work with the dyslexic and his/her family. Schools need to improve the child's motor coordination for dyslexic children are often clumsy. Sobotke et al (1977, p. 368) say perceptual-motor remediation may be good for small children, but it will not remediate reading difficulties with older ones although it might improve handwriting.

Rome and Osman (1979, pp. 671-2) think school curriculum will need to be modified for most dyslexics. Taped assignments, oral examinations, shortened spelling lists, and no foreign languages are some of the requirements. If the school has no small group to help the dyselxics, private tutoring is needed. It is unfair to ask a child to work long hours after school. Remediation may have to be done in the summer. Paine (1972, pp. 77-80) warns that three 20 minute sessions are better than a 1 hour period. He also feels public schools are a better choice than private or parochial ones which stress high academic demands. In some cases, however, a firmly structured program in a parochial school might be beneficial.

Dyslexic students in Denmark come 5 hours per day six days a week. They have 2 hours of reading and writing, 1 hour of arithmetic, 1 hour of music or handicrafts, and 1 hour of some other subject. Night classes are held for adults until 9 p.m. Orton once said that anything which will work is a good method. In Denmark they find that the Norrie phonetic system, the Orton-Gillingham-Stillman technique, and the Borel-Maisonny gestural method all work (Critchley, 1970, pp. 116-17).

According to Rome and Osman (1979, pp. 666-71), Dr. Samuel Orton devised the multi-sensory, three-pathway approach. Later Ann Gillingham worked with him to devise the Orton-Gillingham approach to remediation. Since there are only three sources of learning; visual, auditory, and kinesthetic-tactile, the dyslexic must depend on the latter. A multi-sensory approach uses all the sources of learning. The visual approach may be hard or even impossible for some dyslexics, but the kinesthetic uses speech muscles and hand-arm movements to teach what the student had trouble learning, while the drill methods involve tracing words. Severe dyslexics may need to feel the movements of the mouth, tongue, throat, and lips. A good multi-sensory program involves phonics rules and a structural system moving from simple to complex with much sensory involvement. It avoids an exclusive sight approach to reading, speed reading, rote spelling lists, creeping and crawling, color coding of words, eye-exercises, and inflexible grouping or labeling of children.

Miller (1971, pp. 105-6) says the Fernald tracing method emphasizes the teaching of words through the tracing of syllables. The student traces the word with his/her finger, says the word, and writes it. This process

is repeated until the word is correctly written. Then it is copied on a card for use in a story. It is better for those who need a kinesthetic approach because it uses syllables and does not distort the sounds when blending "cat" to sound like "kuh-a-tuh."

Miller also likes the Gillingham-Stillman method because it uses all the dyslexic's senses. All phonics must be over-learned, for dyslexics need a slow process with much drill. This method teaches words through tracing single letters. The child blends these letters to form words which he/she writes later from memory. There is a danger that the blending can distort the sounds and "kuh-a-tuh" could result.

Clarke (1973, pp. 154-6) notes Miss Norrie in Denmark thinks the brain not the eyes learn to read. Under the Norrie method, sounds are divided according to those produced by the lips, on the front of the tongue, and on the back of the tongue. Miss Norrie uses only sentences that make sense and insists on praising the student at the end of each lesson because any effort the child made deserves praise. The Norrie approach is very similar to the AVT method used in Andrews University's Reading Center.

Miles (1970, pp. 19-41) offers a rather complete method of multi-sensory therapy. He urges the teacher to be flexible and to praise each success while overlooking the mistakes. Miles uses regular words and lets the student read what he/she has written. He says therapy sessions should include 30 minutes on phonics, 10 minutes on straight reading, and 10 minutes on general discussion; thus his sessions are a bit longer than Paine's suggested 20 minutes, but Miles does vary the activities. He also urges the teacher to find interesting material on the child's reading level. If the

child knows the alphabet, making a dictionary is appropriate. The first page involves five columns with a short vowel at the top of each one. The child lists 3-letter words using the appropriate vowel in each column. Page two might have the long vowels with the silent "e," page three the homophones like "sea-see," page four the "oi-oa" words, and page five the irregular words like "father." The child could later use these words for spelling. While this is only one method of remediation, it is a good one.

In summary, one may say that the most effective approach to remediation of dyslexics is a multi-sensory one using a combination of visual, auditory, and kinesthetic-tactile methods. The most famous methods are the Fernald, Orton-Gillingham-Stillman, and the Norrie. Each have their own strengths, and the wise teacher will pick the approach best suited to his/her pupil. The Norrie method is very much like the AVT approach used at Andrews University. A patient, hopeful, and persistent teacher is the most vital ingredient in the dyslexic's learning program.

V. CONCLUSION

The purpose of this study was to learn the meaning of dyslexia, to recognize some of its symptoms, to learn some of its possible causes, and to discover some methods for its remediation. Chapter one has shown that dyslexia means "distorted words." It is the inability to read either phonetically or visually, or both. Among the symptoms of dyslexia in chapter two are motion sickness, a variety of aches to avoid the frustrations of attending school, normal intelligence, retardation in reading of six months to two years, bizarre spelling, auditory-linguistic processing problems, visual-spatial processing problems, daydreaming, and inattention to directions. In chapter three the guesses concerning the cause of dyslexia ranged from prenatal influences to genetic factors and from brain damage to abnormal brain cells. A variety of malfunctions in the brain, ears, and eyes were also suggested. The most plausible cause appeared to be that which was recently offered by Galaburda who found abnormal cells in the brain. These cells were similar to ones producing epilepsy in some people. Such cells might account for nearly all of the abnormal physical problems associated with dyslexia. In chapter four the emphasis was on methods of remediation. Three methods are often used in therapy: the Frenald tracing method uses a sound-syllable approach; the Orton-Gillingham-Stillman method uses a letter-by-letter sound approach; and the Norrie method classifies sounds accoring to where they are formed in the mouth. This latter method is most nearly like the AVT approach at Andrews University. Children learn

through visual, auditory, and kinesthetic-tactile means. A combination of these approaches, a multi-sensory method, is best to use with dyslexics.

In retrospect, very little concerning dyslexia is certain, but it is surely better to assume that poor readers or poor spellers are dyslexic and give them remediation to cope with their problem than to doom them to struggle unsuccessfully for a lifetime in this literate society.

ADDENDUM

After this paper was written, the University of Miami, Florida, reported a study involving grandparents, parents, and children from dyslexic families. The findings revealed an abnormality of the fifteenth chromosome among those family members suffering from dyslexia. The researchers suspect that this chromosome may be the genetic transmitter of dyslexia within families. Such a possibility is not incompatible with Galaburda's finding of abnormal cells in a dyslexic's brain. If dyslexia is hereditary, it could be carried on an abnormal chromosome and cause abnormal cells to form in the brain. The evidence continues to mount that dyslexia is an inherited physical defect.

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